INCIDENCE AND CLINICAL SIGNIFICANCE OF SEVERE MATERNAL ACIDOSIS. ITS INFLUENCE ON THE FETAL ACID-BASE BALANCE

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At birth, arterial blood from mother and neonate is sampled routinely. Between 1973 and 1978, 4,509 out of 6,001 (75 per cent) cases were investigated. In the remaining cases, sampling either failed or was delayed (our deadline is 5 min after complete birth), or was not performed due to lack of motivation of the doctor or lack of cooperation of the patient.

Severe maternal acidosis is defined on the basis of one or any combination of the following values : pH $\leqslant$ 7.25, base excess $\leqslant$ -15.0 mEq/l, and/or lactatemia $\geqslant$ 6 m Eq/l.

This definition is arbitrary, because the limits between normal and pathologic ranges are not known.

Seventy of the cases studied reached one or more of these limits (Table I). The incidence was roughly the same, whichever criterion was applied. As a rule, only two of the three criteria were fulfilled. The number of cases fulfilling one, two, or three criteria amounted to 51, 16, and 3, respectively. Of the 70 acidotic mothers 54 were primiparas.

Further analysis was performed in 22 cases in which the pH was lower than 7.25 and complete records were available. These cases were classified according to mode of delivery.

Table I. Incidence of severe maternal acidosis in the series of 4,509 deliveries

CRITERION FOR ACIDOSIS	pH $\leqslant$ 7.25	BE $\leqslant$ -15.0mEq/l	LACTATE $\geqslant$ 6mEq/l
Number of cases	27	34	31
Percentage	0.60	0.76	0.69

The first noteworthy item is the high proportion of cesarean sections, i.e., 7 out of 22, or one-third of the cases (Table II). One cesarean section was performed before labor started. In the others the patient had been in labor from 1 to 12 h. There was one neonatal death in this group, a preterm baby weighing 1,600 g. The operation was performed because of placenta previa. Autopsy showed hyaline membrane disease. In all cases general anesthesia appeared to be the probable cause of the maternal acidosis. In the mother, the PO_2 was normal or high, as is to be expected when oxygen or a mixture of oxygen and nitrous oxyde is administered. However, PCO_2 was elevated in all instances, which points to marked hypoventilation. Besides respiratory acidosis, there was also metabolic acidosis although the lactate and pyruvate levels were only moderately elevated. In the five cases in which no fetal complications were diagnosed, mixed (respiratory and metabolic) acidosis was found in the neonate in association with only a moderately positive or no fetal-maternal difference. In two cases the indication for cesarean section was severe fetal distress.

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Both of these neonates were severely acidotic (pH_f 7.10 and 6.82).

Except for one case of eclampsia, the course of the 15 vaginal deliveries (Table III) was unremarkable and we are at a loss to explain the etiology of the maternal acidosis. It should be noted that there were no cases of prolonged labor. All but two of the women had been given an intravenous infusion of 5% glucose in water. In the neonates all elements of the acid-base balance were modified to the same extent as in the mother, i.e., the pH and base-excess were lowered, the PCO_2 was elevated but the fetal-maternal differences were normal, and fetal excess lactate in particular was not elevated. In contrast to the cesarean section group, most of the maternal levels of lactate and pyruvate were markedly elevated. The one case of neonatal death was due to periventricular leukomalachia.

If maternal and fetal blood samples are properly collected, i.e., are indeed arterial, the relationship between the fetal and the maternal acid-base balance is quite clear. In clinically normal deliveries metabolic acidosis is higher in the mother than in the fetus. This principle holds for lactate, pyruvate, standard bicarbonate, and base excess. In addition, maternal and fetal lactate and pyruvate levels correlate to such an extent (correlation coefficient : +0.9) that an equilibrium is closely approached on both sides of the placental barrier (1-3). Although the present results do not prove the passage of the acid metabolites from the mother to the fetus or vice-versa, they hardly allow any other explanation. Due to this process, which is called diffusion acidosis, maternal acidosis is parallelled by fetal acidosis. Thus, the fetus remains in harmony with its mother. This principle remains valid even when the mother becomes spontaneously severely acidotic.

Exceptionally, general anesthesia may lead to marked maternal acidosis, probably limited to the induction period. Because six of our patients were in labor at the time of the cesarean section, some degree of metabolic acidosis may have been present as well.

Although these are only preliminary data, they permit the conclusion that maternal acidosis plays a role, albeit infrequently, in some cases of fetal morbidity or mortality.

In sum, it may be said that :

a. Severe maternal acidosis can be expected in one to two per cent of deliveries.
b. It can occur during the induction of general anesthesia, probably as a consequence of hypoventilation.
c. In the unanesthetized parturient its etiology remains obscure.
d. The unimpaired fetus seems to be unaffected. This may not be the case, however, when the fetus is already hypoxic and the abnormal perfusion acidosis is superimposed on the hypoxic changes.
e. More research should be done on the maternal acid-base balance during labor and its influence on the fetus.

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Table II. Maternal acidosis. Caesarean section.

Case n°	Duration of labor (h:min)	Dilatation (cm)	Anesthesia	Fetal outcome
1	12:00	8	general	L&W
2	?	7	general	L&W
3	6:04	3	general	L&W
4	1:05	3	general	L&W
5	0	-	general	L(trisomy 21)
6	(4:30)	3	general	L&W
7	6:04	4	general	NND

Table III. Maternal acidosis. Vaginal delivery.

Case n°	Duration of labor 1st stage (h:min)	2nd stage (min)	Anesthesia	Fetal outcome
1	2:30	54	none	L&W
2	>3:39	9	none	L&W
3	13:00	40	CLEA	NND
4	3:20	3	none	L&W
5	6:40	17	none	L&W
6	2:00	23	none	L&W
7	7:00	22	none	L&W L&W(B) L&W(B)
8	6:40	28	none	L&W
9	5:30	13	none	L&W
10	(2:00)	10	none	L&W
11	>9:00	9	none	L&W(B)
12	6:00	13	none	L&W
13	8:10	15	pethidine	L&W
14	10:15	15	CLEA (failed)	L&W
15	1:30	18	none	L&W

L&W : living and well; NND : neonatal death; (B) : breech presentation; CLEA : continuous lumbar epidural analgesia

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